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Overview of Statistics on Acute and Chronic Water Contamination Problems

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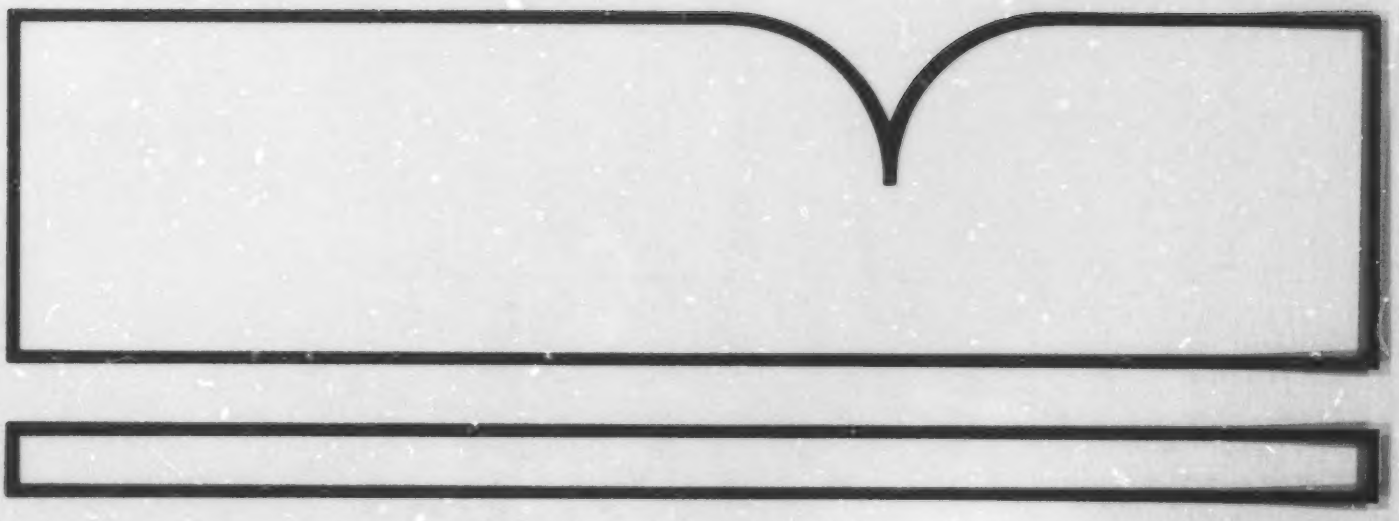

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AN OVERVIEW OF STATISTICS ON ACUTE AND CHRONIC WATER CONTAMINATION PROBLEMS

Gunther F. Craun

Coordinator, Environmental Epidemiology Program

US Environmental Protection Agency

Cincinnati, Ohio

HEALTH EFFECTS RESEARCH LABORATORY
OFFICE OF RESEARCH AND DEVELOPMENT
U.S. ENVIRONMENTAL PROTECTION AGENCY
RESEARCH TRIANGLE PARK, NORTH CAROLINA 27711

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16. ABSTRACT

Discussed in this paper are waterborne outbreaks of acute disease and the association of water contaminants with cardiovascular disease and cancer. A selected list of references provided for those wishing a more in-depth report.

17. KEY WORDS AND DOCUMENT ANALYSIS

a. DESCRIPTORS	b. IDENTIFIERS/OPEN ENDED TERMS	c. COSATI Field/Group

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Introduction

I appreciate the opportunity to discuss with you today an area of research in which I have worked for some 15 years. With the time restrictions of this conference, I have found it necessary to narrow the topic somewhat, and rather than present a broad overview, I will focus on what I consider to be the major health-related concerns: the occurrence of waterborne outbreaks of acute disease and the association of water contaminants with cardiovascular disease and cancer. Most of my comments have been addressed in other articles which are soon to be published elsewhere (Craun, in press). For those of you who wish a more in-depth discussion of these and other areas of concern in the drinking water, I have provided a selected list of references.

Outbreaks of Waterborne Disease

Statistical data have been collected on waterborne outbreaks in the United States since 1920[a], and I have recently summarized these data. The reporting of waterborne outbreaks has been and continues to be voluntary, and all of the information on these outbreaks were obtained from the scientific and medical literature and through the cooperation and assistance of state and local health officials, epidemiologists, and engineers. It is recognized that these data may reflect differences in the surveillance and reporting interests of each of the states over the past 64 years, but it is felt that the data are reasonably complete for an analysis of the causes and etiologies of waterborne outbreaks.

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aNo data were collected in 1937

For this summary, all water systems were classified as community, noncommunity, and individual according to definitions contained in the Safe Drinking Water Act (P.L. 93-523). Community systems are public or investor-owned water supplies serving communities, subdivisions, and mobile-home parks of at least 25 year-round residents or 15 service connections used by year-round residents. Noncommunity systems are seasonally-operated water supplies or supplies that serve travelers and transients. These systems must serve an average of at least 25 individuals for 60 or more days during the year; this includes institutiions, industries, camps, parks, hotels, and service stations which maintain their own water supply for employees and the traveling public. Individual systems are water supplies used by residents in areas without community systems or by persons traveling outside of populated areas (e.g., backpackers, campers).

During 1920 through 1936 the criterion of at least five cases of illness was used to define an outbreak, but during 1938 through 1983 an outbreak was defined as two or more cases of illness. This inconsistency in defining an outbreak is not felt to cause a major problem in intrepreting the data, since only 12 outbreaks of fewer than five cases were reported and excluded from the 1920-36 data base. Single cases of acute chemical poisoning where the chemical was identified in the water system, such as methemoglobinemia caused by high nitrates in water, were also included as outbreaks. In many of the outbreaks, drinking water was found to be bacteriologically or chemically contaminated, but in only a few of the outbreaks was the etiologic agent isolated from drinking water. In all of the reported outbreaks, however, drinking water was

epidemiologically implicated as the vehicle of transmission of the illness. Outbreaks associated with water used or intended for drinking or domestic purposes and outbreaks associated with the use of contaminated springs and creeks by backpackers and campers were classified according to cause of the outbreak. Outbreaks associated with swimming and water not intended for drinking purposes are included under miscellaneous causes when information was made available on these outbreaks.

During 1920 through 1983, 1531 waterborne outbreaks were reported in the United States. A total of 415,329 cases of illness and 1083 deaths were associated with these outbreaks. Each of the 50 states, the District of Columbia, and Puerto Rico reported at least one outbreak. Forty-four percent of these outbreaks occurred in community water systems compared with 35 percent in noncommunity and 21 percent in individual water systems (Table 1). The majority of the cases of illness (85 percent) and deaths (71 percent) were associated with outbreaks in community water systems.

The number of outbreaks reported annually ranged from a minimum of six in 1957 to a maximum of 60 in 1941 with a mean of 24.3 and a median of 22.9 outbreaks for the entire period (Figure 1). Waterborne outbreaks reported from 1920 through 1930 were uniformly distributed with an mean of 23 outbreaks. Thereafter, the reporting of waterborne outbreaks was cyclical with more than 40 outbreaks being reported annually during 1938 through 1941 and 1979, 1980, 1981 and 1983 and less than 20 outbreaks reported annually during 1950 through 1970. Because the reporting of waterborne outbreaks is voluntary, these flucuations must be largely attributed to differences in the capabilities and interests of health

agencies to investigate and report waterborne outbreaks over these years.

During 1920 through 1936, waterborne outbreaks were reported most frequently in community water systems; afterwards outbreaks were generally reported most frequently in noncommunity water systems. A distinct seasonal occurrence of waterborne outbreaks was observed for noncommunity and individual water sytems but not for community water systems. Outbreaks in noncommunity water systems occurred most frequently during June through August, and outbreaks in individual water systems occurred most frequently during June through September. Outbreaks in noncommunity systems effect primarily travelers, campers, restaurant patrons, and visitors to recreational areas, and the seasonal distribution of waterborne outbreaks may be due to either increased contamination of water supplies during the summer or an increased number of susceptible individuals using water supplies which are always contaminated.

Cases of illness caused by waterborne outbreaks ranged from a minimum of 76 in 1955 to a maximum of 45,992 in 1926 with a mean of 6,592.5 and a median of 3,611 cases (Figure 2). Although the reported number of cases vary from year to year, several trends can be observed. The the number of illnesses generally declined from 1940 through 1955 and increased from 1955 through 1983. In 1980 and 1984, some 20,045 and 21,124 cases of illness were reported, the latter being the largest number of cases of waterborne illness reported since 1940. Despite the recent increase in reported waterborne outbreaks and cases of waterborne illness, the incidence of waterborne disease has decreased from approximately eight cases of waterborne disease per 100,000 person-years in 1920-

40 to approximately four cases per 100,000 person years in 1971-83.

Approximately one-half of the waterborne outbreaks resulted in fewer than 26 cases of illness, and over 75 percent of the outbreaks resulted in fewer than 100 cases of illness (Table 2). Only four percent of the outbreaks resulted in more than 1000 illnesses. An average of 369 cases of illness per outbreak occurred during 1920-40, compared with 157 cases of illness per outbreak during 1941-60 and 266 cases of illness per outbreak during 1961-83. However, the magnitude of a waterborne outbreak depends primarily upon the type of water system involved, since large outbreaks in community water systems can greatly influence these data. In general, outbreaks in community water systems have resulted in many more cases of illness per outbreak than noncommunity systems and individual systems. For example during 1971-80, community water systems averaged 480 cases of illness per outbreak compared with 116 cases per outbreak in noncommunity systems and 10 cases per outbreak in individual systems.

An etiologic agent was determined in 56 percent of the waterborne outbreaks (Table 3). The remaining outbreaks resulted in acute gastroenteritis characterized by symptoms including abdominal cramps, nausea, vomiting, and diarrhea occurring 12 to 48 hours after the consumption of contaminated water. It is difficult to estimate how many of the gastroenteritis outbreaks were caused by organisms which are commonly transmitted by water but were not identified because clinical specimens were either not collected or not collected in a timely manner. In many of the outbreaks, the search for an etiologic agent was limited to those organisms easily cultured at the time, and the etiologic agent was not isolated

because the appropriate laboratory analysis was not conducted or not available at the time. These outbreaks could have been caused by commonly transmitted waterborne agents or unrecognized, less frequently transmitted waterborne agents. In recent years waterborne viral gastroenteritis has been reported with increasing frequency because of the availability of more sensitive laboratory techniques for identification of viral agents. It is possible that viral agents, as well as some of the newly recognized bacterial agents (toxigenic Escherichia coli, Campylobacter fetus, Yersinia enterocolotica), have been important unrecognized causes of waterborne outbreaks of gastroenteritis of undetermined etiology. However, in some of the largest outbreaks occurring in recent years, an etiology could not be established through extensive laboratory analysis of both human specimens and water samples including appropriate tests for newly recognized bacterial and viral pathogens, parasites, and chemicals. This suggests that additional unrecognized agents may be responsible for waterborne gastroenteritis.

The diseases most frequently transmitted by contaminated drinking water in the United States were acute gastroenteritis of undetermined etiology, typhoid fever, shigellosis, hepatitis A, giardiasis, and acute chemical poisonings. The pathogen most commonly identified in waterborne outbreaks prior to 1960 was Salmonella typhi. During 1961 through 1983 Giardia lamblia, hepatitis A, and Shigella were the most commonly identified pathogens. Although waterborne outbreaks of salmonellosis, tularemia, amebiasis, para-typhoid fever, leptospirosis and poliomyelitis have been reported, the transmission of these diseases in the United States by consumption of contaminated water appears to be of minor importance.

There were 1083 deaths associated with waterborne outbreaks. The majority of these deaths were due to typhoid fever and occurred early in the period of study (Table 3). Eighty-four percent of the deaths occurred prior to 1936, and less than one percent of the deaths occurred after 1970. Almost all deaths due to typhoid fever (94 percent) and amebiasis (96 percent) occurred prior to 1941. The primary causes of death in recent outbreaks were shigellosis and acute chemical poisonings.

The major cause of waterborne outbreaks (43 percent) was the use of contaminated, untreated groundwater (Table 4). However, the greatest number of cases of waterborne illness (37 percent) resulted from outbreaks caused by deficiencies in the treatment of surface water sources (Table 5). Deficiencies in the treatment of all water sources were responsible for 333 outbreaks (21 percent) while deficiencies in the distribution of water were reponsible for 233 outbreaks (16 percent). Although use of contaminated, untreated surface water caused 158 outbreaks (10 percent), this is largely a problem of the past, since only a few small water systems and a small number of individuals currently use surface water supplies with no treatment.

The use of contaminated, untreated groundwater and the inaedequate or interrupted treatment of groundwater sources caused some 51 percent of the outbreaks and 36 percent of the cases of illness. The overflow or seepage of sewage and surface runoff were responsible for 40 percent of the waterborne outbreaks which occurred as the result of using contaminated, untreated springs; data were insufficient to determine the source of contamination of springs in approximately half of the outbreaks. A

source of contamination was identified in 76 percent of the waterborne outbreaks caused by contaminated, untreated well water. The overflow or seepage of sewage into wells caused 35 percent of these outbreaks; surface runoff into wells from heavy rains or flooding caused 21 percent of these outbreaks; and contamination of wells through creviced limestone or fissured rock caused 8 percent of these outbreaks. Other sources of contamination of wells included chemicals, contaminated surface water from streams or rivers, seepage from abandoned wells, and animals entering the well. Examination of the etiologies over the most recent 13 year period showed chemicals, Shigella, and hepatitis A to be the most important etiologic agents of outbreaks in untreated well water systems.

Twenty waterborne outbreaks occurred after unexpected heavy contamination overwhelmed the normal disinfection requirements. Most of these outbreaks occurred in surface water systems where contamination was caused by the overflow or seepage of sewage. Inadequate or interrupted disinfection of both surface and groundwater resulted in 268 waterborne outbreaks. In groundwater systems, interrupted disinfection was a greater cause of outbreaks than inadequate disinfection. Conversely, in surface water systems inadequate disinfection caused more outbreaks than interrupted disinfection. Inadequate control of filtration and allied treatment was responsible for only a small number of the outbreaks caused by treatment deficiencies but was responsible for a large number of the cases of illness, since large water systems were often involved.

Cross-connections, including backsiphonage, were responsible for the majority of outbreaks and illnesses caused by contamination in the distribution system. Most of the outbreaks caused by cross-connections

resulted in gastroenteritis of unidentified etiology, typhoid fever, and acute chemical poisoning. Outbreaks were also caused by contamination of distribution reservoirs in community water systems, contamination of water mains during repair or construction and corrosion problems in the distribution system.

Waterborne outbreaks classified as having miscellaneous causes include outbreaks resulting from the use of water not intended for drinking, contaminated ice, buckets, hoses, tank trucks, and drinking fountains, consumption of water during swimming, and suspected deliberate contamination with chemicals.

Sixty outbreaks and 3924 cases of illness occurred as the result of acute chemical poisonings caused primarily by high concentrations of copper, fluoride, arsenic, petroleum products, and pesticides in water (Table 6). The outbreaks ranged in size from single cases of infantile methemoglobinemia caused by consumption of high nitrates in well water to a large outbreak of several thousand cases of chlordane poisoning in Pittsburgh in 1980. Most of these outbreaks were caused by contamination of groundwater, contamination through cross-connections, or corrosive water leaching metals from plumbing materials. The eight outbreaks of acute fluoride poisoning were caused by improperly designed or malfunctioning fluoridation equipment and operational problems in the addition of fluoride. Five of these outbreaks occurred at elementary schools adding fluoride to their own water system. These incidents illustrate the need for improved design of fluoridation equipment, increased surveillance, and operator training.

The development of infantile methemoglobinemia from nitrate in drinking water depends upon the bacterial conversion of nitrate to nitrite before or after ingestion. This occurs almost exclusively in infants less than three months old. The three cases of methemoglobinemia reported here do not reflect the actual incidence of this disease over the study period. Methemoglobinemia is not a reportable disease in the United States, and only recently has this disease been included in the statistics on waterborne outbreaks.

Nitrate in drinking water was first associated with infantile methemoglobinemia in 1945. Since then, approximately 2000 cases have been reported in North America and Europe with about eight percent of these cases resulting in death. Almost all of the 335 cases occurring in the United States were reported in the 1940's and 1950's. Since 1960, only one case associated with high nitrates in a community water system and only two additional cases of infantile methemoglobinemia associated with high nitrates in individual well water systems have been described in the medical literature. However, studies of hospital records revealed other cases of infantile methemoglobinemia: one case associated with high nitrates in individual water supplies in Nebraska during 1973-79 and ten cases in Oklahoma during 1960-71. A survey of 37 states in 1979 revealed only two additional reported cases associated with nitrates in individual wells over the previous ten years. Although cases of infantile methemoglobinemia associated with nitrate in water continue to be reported more routinely in eastern Europe, the sporatic incidence of this disease in the United States suggests that other factors may be more important for the development of methemoglobinemia in this country. It is probable

that changes in infant feeding practices (e.g. increased breast feeding and decreased use of powdered formula) and health education in high nitrate areas have reduced exposure of infants to high nitrate water.

Water Quality and Cardiovascular Disease

Since 1957 when Kobayashi first reported a statistical correlation between the acidity of water supplies in Japan and cerebrovascular disease mortality, numerous investigators have reported an association between drinking water quality and cardiovascular disease. In general, epidemiologic studies have shown an inverse association between water hardness and cardiovascular disease mortality; that is, lower cardiovascular mortality is found in areas where the hardness of drinking water is high. In areas of the United Kingdom where drinking water supplies are extremely soft, cardiovascular disease mortality is about 30 percent higher than in areas where drinking water supplies are extremely hard. In the United States a difference in cardiovascular disease mortality of about 15 percent is found between areas with soft and hard water supplies.

At the present time, the association between water hardness and cardiovascular disease must be considered to be statistical rather than causal. Although biologically plausible in some respects, the epidemiologic evidence is not specific. Individual exposures and possible confounding by individual risk factors have only been considered in a few recent studies, and the observed associations have not been strong. Altitude, climate, latitude, humidity, air temperature, and socio-economic status have also been found to influence the association between water hardness and cardiovascular disease mortality. In addition, water hardness has

been found to be associated with other causes of death. While the inverse association of water hardness and cardiovascular disease mortality was reported in most, but not all, studies involving large areas, attempts to confirm these findings in small areas have generally not been successful.

Findings from the British Regional Heart Study, which was initiated several years ago to obtain information about the possible causes of cardiovascular disease and the regional variations in ischemic heart disease and stroke in the United Kingdom, indicate that the association of water hardness and cardiovascular disease mortality cannot be entirely explained by other environmental, social, or personal risk factors. The evidence suggests that areas in the United Kingdom with very soft water supplies may have a ten percent excess cardiovascular disease mortality after allowance for climatic and social factors. Sharett et al. (1979) analyzed data from 484 cities in the United States and after adjustment for various demographic variables, found cardiovascular disease mortality rates to be approximately five percent higher in cities using soft water compared with cities using hard water in the same region. After controlling for climate and socio-economic factors in 234 British towns, Shaper et al. (1980) found a three to four percent increase in cardiovascular disease mortality for every 100 mg/l decrease in total water hardness. A clinical study of 7735 British men also suggested an association between water hardness and nonfatal ischemic heart disease with an effect observed only for water hardness of less than 170 mg/l total hardness. These men will continue to be studied for both fatal and nonfatal cardiovascular events.

Zeighami et al. (in press) recently reported results of a case-comparison epidemiologic study of 505 white male Wisconsin farmers who had died from coronary artery disease or cerebrovascular disease and 854 living comparison farmers without evidence of cardiovascular disease. All farmers obtained drinking water from individual unchlorinated wells. The drinking water of farmers who had died of cardiovascular disease was found to be softer than that of the comparison group. The association of cardiovascular disease mortality and calcium in drinking water was more consistent than the association of several other water parameters and cardiovascular disease mortality. Similar to the findings of the British Regional Heart Study, an effect was noted only for water hardness of less than 200 mg/l. Evidence exists for an association between sodium intake and blood pressure and some studies have suggested that cardiovascular disease may also be related to sodium intake. However, this study offered no evidence of a relationship between coronary artery disease or cerebrovascular disease and sodium in drinking water. Two recent reports have suggested that chloride rather than sodium may be responsible for increased blood pressure in experimental animals.

If water hardness does indeed play a role in the development of cardiovascular disease, hard water could be beneficial by preventing a deficiency of an essential element(s) or soft water could be detrimental by contributing an excess of a toxic element(s). Calcium and magnesium may likely be the beneficial constituents of hard water, since they have been found to play a role in the development of hypertension and other adverse effects on the cardiovascular system in experimental animals. Epidemiologic studies have shown an association between high blood pres-

sure and decreased calcium intake, and clinical studies have shown that increasing calcium intake has lowered blood pressure. Increased calcium intake has also been shown to attenuate a sodium-induced blood pressure increase in rats. Magnesium is important for myocardium function, and low levels of magnesium in tissues appear to occur more often in soft water areas. Rats on diets moderately or severely deficient in magnesium showed significant elevations in blood pressure. The intake of both calcium and magnesium may be below recommended values for a substantial portion of the population, and hard water can be an important nutritional source for both elements, providing from 10 - 50 percent of the usual daily intake. In addition, high calcium levels in hard water may reduce or prevent the absorption of toxic metals, such as lead and cadmium, both of which have been associated with the development of hypertension in experimental animals. Most clinical effects of toxic metals appear only in animals in a calcium deficient diet.

In general, soft waters are more corrosive than hard waters and may contain higher levels of metals leached from water pipes and plumbing materials. However, characteristics other than hardness are important in determining the corrositivity of drinking water. Reducing the corrositivity of drinking water can prevent the leaching of metals from plumbing materials, but it is not clear at this time whether this will also result in decreased cardiovascular mortality.

Another consideration is the potential effect of sodium which may be added to drinking water when hard water is artificially softened by ion exchange. The resulting levels of sodium could adversely effect those

individuals on low-sodium diets. In addition, several recent epidemiologic studies have reported a slight increase in blood pressure in children and young adults associated with high sodium concentrations in water, but these studies should be interpreted with caution until better evidence is provided on the magnitude of this effect.

In summary, additional evidence has been obtained in recent years to further suport the association between water hardness and cardiovascular disease, but more research is required before recommendations can be made regarding the advisability of removing calcium or magnesium from drinking water by either ion exchange or lime-soda ash softening. Pocock et al. (in press) emphasize it is important to recognize that certain personal risk factors have a much greater influence on health. Changes in smoking behavior and adoption of a prudent diet may offer far greater rewards in the prevention of cardiovascular disease than changes in water hardness.

Chlorination Of Drinking Water And Cancer

The use of chlorine as a disinfectant to prevent the waterborne transmission of infectious diseases has been implicated as contributing organic micropollutants to drinking water. Under certain conditions, the reaction of free chlorine with selected precursor substances in drinking water produces trihalomethanes (THMs), a group of halogen-substituted single carbon compounds. The predominant chlorination by-products are chloroform and bromodichloromethane, but dibromochloromethane and bromoform are also frequently found. The concentration of the THMs is dependent upon the presence and concentration of the precursors, chlorine dose

and contact time, water temperature, and pH. Under typical circumstances chloroform is the compound most often formed and is found at concentrations generally exceeding those of other organic micropollutants by a substantial margin. Chloroform has been shown to be a carcinogen in animal studies at high dose levels, and the other THMs have been found to be mutagenic in bacterial tests. Since the important percursors which react with free chlorine to produce these compounds are naturally occurring aquatic humic substances, such as humic and fulvic acids and bromide, chloroform and the other THMs have likely been present in water supplies as long as chlorine has been used as disinfectant.

Since 1974 a number of statistical and epidemiologic studies have been conducted in the United States to assess the relationship between cancer and drinking water quality. These studies differ markedly in their design and in what they can reveal about these possible relationships. Because of the widespread exposure to chlorination by-products, most of these studies have focused on the relationship between cancer and the chlorination of drinking water. Historical information on water sources and chlorination practices have primarily been used as surrogate measures of exposure to THMs, but in some instances current levels of THMs have been used in conjunction with various operational parameters to estimate historic exposure levels. Many of the early studies were descriptive in design, and information is available either only on exposure and disease for groups of persons or only on disease. The epidemiology subcommittee of the National Academy of Sciences Safe Drinking Water Committee reviewed all of these descriptive studies conducted through 1978. Nine of ten descriptive studies found associations between chlori-

nated surface water and either cancer incidence or mortality, and three additional studies found associations between current levels of THM and cancer mortality. These studies are felt to be of limited value, as doubts remain as to whether the observed associations are a result of exposure to chlorinated water or due to potential confounding characteristics. Although some demographic variables were considered in most of these studies, data are not available for individuals, and it is impossible to adequately control for the effects of potential confounders. The National Academy of Sciences' review noted that "the bladder, stomach, large intestine and rectum, which were cancer sites identified in a number of geographic areas, warrant further study" and recommended analytic epidemiologic studies be conducted so a quantitative measure of the association could be obtained. In an analytic study, information on exposures, disease, and potential confounding characteristics are available for each study participant, and confounding bias can be assessed and controlled. A quantative statement about an observed association between water chlorination and cancer can be made using the data from analytic studies.

Recently, results have been reported for several analytic studies of the relationship between water chlorination and cancer in the United States: a cohort study and several case-comparison studies. Wilkins and Comstock (1981) studied three historical cohorts, each distinguished by a different degree of exposure to chloroform and other chlorination by-products. Incidence rates for cancer of the bladder among men and cancer of the liver among women were found to be nearly two-fold higher in the cohort supplied with chlorinated surface water at home when compared with

the cohort supplied with unchlorinated groundwater. The results of this study cannot be interpreted as reliable evidence of an association because few cancer deaths occurred. Thus, the reported rates are unstable and sensitive to random variation.

A variation of the traditional case-comparison study using data available from death certificates for decedent cases and comparison subjects has been used by several investigators to obtain information on associations of cancer and water quality. Since no interviews are conducted in this type study, the advantages of this approach include considerable cost savings, but the disadvantages of this approach include the loss of important information on residence history, exposures, and confounding factors. A review (Crump and Guess, 1982) of five case-comparison mortality studies completed through 1981 concluded that information provided by the studies strengthened the evidence for an association between rectal, colon, and bladder cancer and water chlorination but was not sufficient to establish a causal relationship between chlorinated by-products in drinking water and cancer. Because of methodological limitations and the small increased risks observed in these studies, it is not possible to separate possible associations from potential confounding characteristics which could not be assessed or controlled.

The two most recently reported studies have employed traditional case-comparison study designs where individuals were interviewed (Cragle et al., 1985; Cantor et al., 1985). Cragle et al. investigated the relationship between water chlorination and colon cancer using 200 incident cases of colon cancer from seven hospitals in North Carolina and 407 hospital-based

comparison subjects without evidence of cancer, familiar polyposis, ulcerative colitis, adenomatus polyposis, or any major chronic intestinal disorder. Both cases and comparison subjects were residents of the state for at least ten years. Comparison subjects were matched on age, race, gender, vital status, and hospital. Additional information on various potential confounders including alcohol consumption, genetic risk (number of first-degree relatives with cancer), diet, geographic region, urbanicity, education, and number of pregnancies, was obtained by either mail questionnaire or telephone interview. Approximately 71 percent of the eligible population participated in the study. Water exposures were verified for each address and categorized as chlorinated and nonchlorinated for the analysis. Logistic regression analysis found several factors to be positively associated with colon cancer: genetic risk, an interaction term between alcohol consumption and high fat diet, and an interaction term between age and chlorination. The relationship between chlorinated water and colon cancer was found to be dependent upon age and years of exposure. Relative risks for persons who drank chlorinated water at their residence for 16 or more years were consistently higher than those exposed to chlorinated water less than 16 years, but a statistically significant association between water chlorination and colon cancer, controlling for possible confounders, was found only for those above age 60 (Table 7).

Cantor et al. recently reported results from a collaborative EPA-National Cancer Institute (NCI) study of the association between water chlorination and bladder cancer. Included were a total of 2982 persons (73 percent of those eligible to participate) between the ages of 21 and 84 diagnosed with cancer of the urinary bladder in 1978 and residing in ten

areas of the United States (Connecticut, Iowa, New Jersey, New Mexico, Utah, and the metropolitan areas of Atlanta, Detroit, New Orleans, San Francisco, and Seattle) and 5782 population-based comparison subjects, randomly selected and frequency matched on gender, age, and study area. Subjects were interviewed at home by a trained interviewer, and data were collected for a number of possible confounders including smoking, occupation, artificial sweetner use, coffee and tea consumption, and use of hair dyes. A complete residence history was obtained to categorize individuals according to water sources and chlorination status on a year by year basis, and information was obtained on use of bottled water and fluid consumption. Of the 587,568 person-years lived by all residents since 1940, 76 percent were at a known water source. Logistic regression analysis was used to control for potential confounders. Among persons in all study areas combined, relative risk was not elevated in those participants living in areas with chlorinated water supplies. However, it should be noted that this study was originally designed to determine if saccharin was a human carcinogen rather than to determine cancer risks associated with water chlorination. The study areas were not selected to provide the optimal variabilty of water sources and treatment, and the statistical power of the study is less than suggested by the large number of individuals studied, as the five metropolitan areas are served primarily by chlorinated water supplies. Among the 10 study areas, participants from the three states with agricultural land use did show elevated risk for bladder cancer as the number of years at a surface source increased, but the number of participants in these areas was small compared with the other areas. Among nonsmokers who were never employed in a high-risk occupation (a group otherwise at low

risk for bladder cancer), the risk was elevated among those served for 60 or more years by chlorinated surface sources. (Table 8).

To further study the suggestion of an association between chlorinated water and bladder cancer incidence in agricultural areas, a collaborative case-comparison study is being planned with NCI to begin in late 1985. Approximately 2500 incident cases of cancer of the colon, rectum, bladder, brain and pancreas (liver and kidney if feasible) and approximately 1500 population-based controls matched for race, gender, age will be interviewed for information on lifetime residential history, smoking, occupation, medical history, diet, socio-economic status to investigate cancer risks associated with chlorinated drinking water and agricultural runoff. An exposure history will be developed from the lifetime residential history.

Young et al. (1981) reported an association between colon cancer mortality in Wisconsin and THMs exposures in drinking water as estimated by the average daily chlorine dosage of water over a twenty-year period. The study included 8,029 cancer deaths and 8,029 noncancer deaths in white females matched on county of residence, year of death, and age. Death certificates provided information on urbanicity, maritial status, and occupation, and these were considered as potential confounders. This association was further pursued in an interview study of 370 incident cases of colon cancer and 1450 population-based comparison subjects. This case-comparison study found no association between estimated THMs exposures and colon cancer incidence, but colon cancer cases were found to more frequently consume water from municipal groundwater supplies. Since groundwaters in the United States have been shown to be contaminated with synthetic votalite

organic compounds the possibility must be considered that misclassification of exposure occurred and resulted in the lack of an association observed in this study. A study to examine the exposures of this population to organic contaminants in groundwater is planned. The data obtained from this epidemiology study will be reanalyzed considering these exposures.

In summary, the two most recently reported case-comparison epidemiologic studies have provided an indication of the magnitude of the cancer risk associated with chlorinated water for colon and bladder cancer. A weak to moderate association between water chlorination and colon cancer was observed in an elderly population, and this association was found to be stronger among the elderly who had been exposed to chlorinated water for more than 15 years. A moderately strong association between chlorinated water and bladder cancer was observed in an otherwise low-risk population of nonsmokers, who had received chlorinated surface water for 60 or more years. Additional case-comparison or cohort studies are required to confirm these findings and are currently being planned. Any interpretation as to the causal association suggested by these studies should await the completion of these planned studies and other relevant research in this area.

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Table 1

Waterborne Outbreaks in the United States
1920-83

Type of Water System	Number of Outbreaks	Cases of Illness	Deaths
Community	669	351,333	767
Noncommunity	536	56,639	92
Individual	326	7,357	224
Total	1,531	415,329	1,083

Table 2

Magnitude of Waterborne Outbreaks
1920-83

Size of Outbreak (Cases of Illness)	Frequency of Occurrence	
	Number of Outbreaks	Percent of Outbreaks of Smaller Size
<2	7	<1%
2-5	165	11%
6-10	218	25%
11-25	355	49%
26-50	274	67%
51-100	190	79%
101-200	127	87%
201-300	45	90%
301-500	53	94%
501-1000	43	96%
1001-3000	33	99%
3001-5000	10	99+%
5001-10,000	5	99+%
>10,000	6	100%

Table 3

Etiology of Waterborne Outbreaks
1920-83

Time Period	Disease	Number of Outbreaks	Cases of Illness	Deaths
1920-40	Typhoid Fever	372	13,761	889
	Gastroenteritis	144	176,725	2
	Shigellosis	10	3,308	0
	Amebiasis	2	1,416	98
	Hepatitis A	1	28	0
	Chemical Poisoning	1	92	0
	TOTAL	530	195,330	989
1941-60	Gastroenteritis	265	54,439	3
	Typhoid Fever	94	1,945	54
	Shigellosis	25	8,951	8
	Hepatitis A	23	930	0
	Salmonellosis	4	31	0
	Chemical Poisoning	4	44	4
	Para-Typhoid Fever	3	19	0
	Amebiasis	2	36	2
	Tularemia	2	6	0
	Leptospirosis	1	9	0
	Poliomelitis	1	16	0
	TOTAL	424	66,426	71
1961-83	Gastroenteritis	266	86,740	0
	Giardiasis	84	22,897	0
	Chemical Poisoning	55	3,788	7
	Shigellosis	52	7,462	6
	Hepatitis A	51	1,626	1
	Typhoid Fever	19	330	0
	Salmonellosis	17	18,951	3
	Viral Gastroenteritis (Norwalk)	16	3,973	0
	Campylobacteriosis	5	4,773	0
	Toxigenic *E. coli* Gastroenteritis	5	1,188	4
	Amebiasis	3	39	2
	Viral Gastroenteritis (rotavirus)	1	1,761	0
	Cholera	1	17	0
	Yersiniosis	1	16	0
	Para-Typhoid Fever	1	5	0
	TOTAL	577	153,566	23

Table 4

Causes of Waterborne Outbreaks
1920-83

Cause	Outbreaks	
	Number	Percent
1. Use of Contaminated, Untreated Groundwater	661	43%
2. Treatment Deficiencies in Surface Water Systems	187	12%
3. Use of Contaminated, Untreated Surface Water	158	10%
4. Cross-Connections	148	10%
5. Miscellaneous Causes and Insufficient Data	146	10%
6. Treatment Deficiencies in Ground-Water Systems	126	8%
7. Corrosion Problems and Contamination of Mains, Plumbing and Storage Facilities Other Than Through Cross-Connections	85	6%
8. Treatment Deficiencies in Systems With Combined or Unspecified Water Sources	20	1%
TOTALS	1,531	100%

Table 5

Causes of Illness in Waterborne Outbreaks
1920-83

Cause	Cases of Illness	
	Number	Percent
1. Treatment Deficiencies in Surface Water Systems	153,464	37%
2. Use of Contaminated, Untreated Groundwater	82,528	20%
3. Cross-Connections	68,567	17%
4. Treatment Deficiencies in Ground-Water Systems	65,428	16%
5. Corrosion Problems and Contamination of Mains, Plumbing and Storage Facilities Other Than Through Cross-Connections	15,010	4%
6. Miscellaneous Causes and Insufficient Data	11,542	3%
7. Use of Contaminated, Untreated Surface Water	12,709	3%
8. Treatment Deficiencies in Systems With Combined or Unspecified Water Sources	6,081	<1%
TOTALS	415,329	100%

Table 6

Chemicals Responsible for Multiple Outbreaks of Waterborne Disease
1939-83

Chemical	Outbreaks	Illnesses
Copper	10	120
Fluoride	8	664
Arsenic	6	37
Petroleum products	5	111
Chlordane	4	2,043
Unidentified pesticides	4	22
Nitrate	3	3
Lead	2	89
Chromates	2	31
Selenium	2	7
Herbicides	2	5

Table 7

Chlorination Odds Ratios for Colon Cancer Reported by Cragle et al. (1985) in the North Carolina Study

Age	1-15 years exposure Relative Risk (95% C.I.)	>15 years exposure Relative Risk (95% C.I.)
60-69	1.18 (0.94, 1.47)	1.38 (1.10, 1.72)
70-79	1.47 (1.16, 1.84)	2.15 (1.70, 2.69)
80-89	1.83 (1.32, 2.53)	3.36 (2.41, 4.61)

Table 8

Relative Risks for Bladder Cancer in Nonsmokers Served by a Chlorinated Surface Water Source (Cantor et al., 1985)

Years at a Residence Served by Chlorinated Water	Relative Risk (95% C.I.)
1-19	1.3 (0.7, 2.2)
20-39	1.5 (0.9, 2.4)
40-59	1.4 (0.9, 2.3)
60+	2.3 (1.3, 4.2)

FIGURE 1

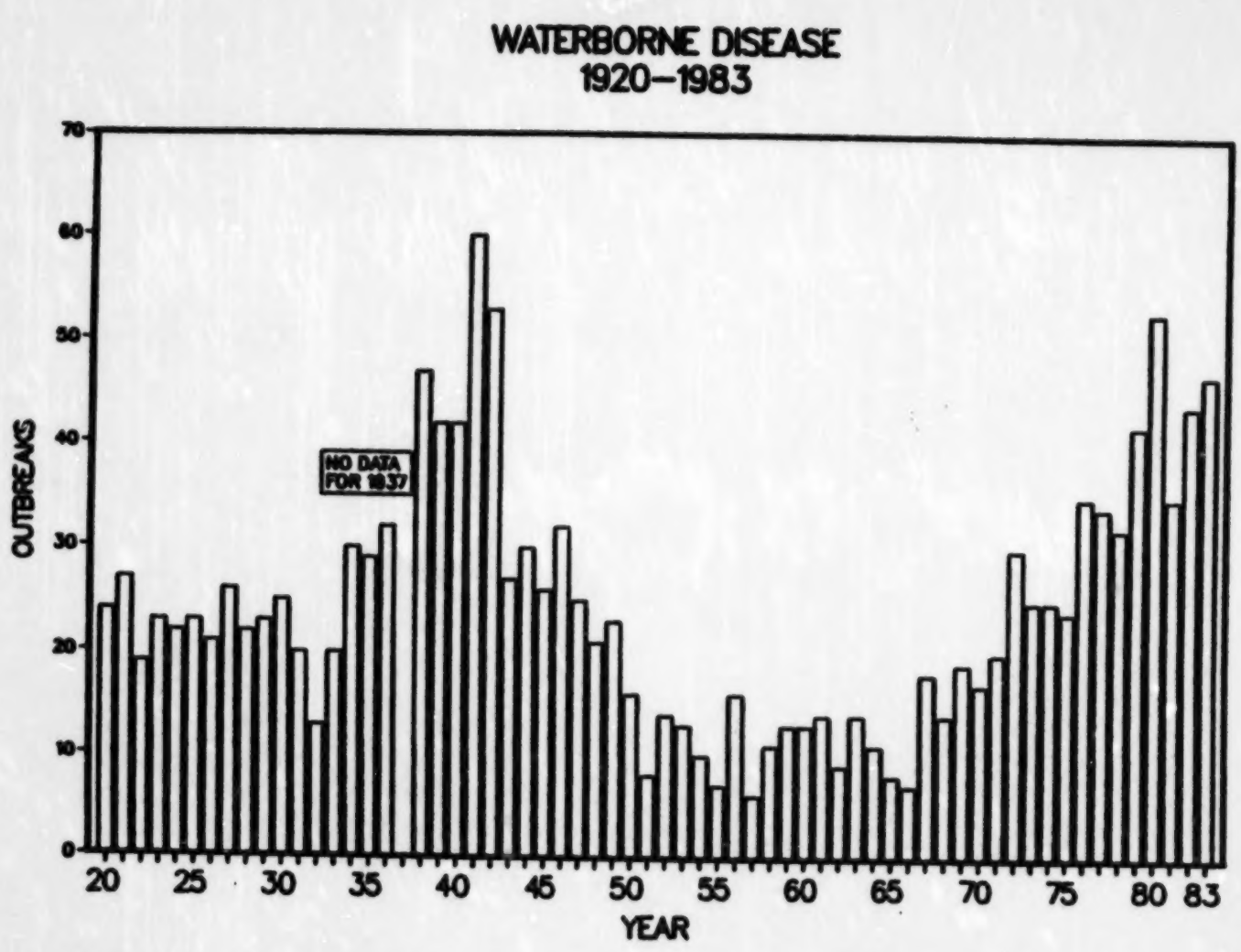
WATERBORNE DISEASE
1920–1983
OUTBREAKS
0
10
20
30
40
50
60
70
NO DATA FOR 1937
20
25
30
35
40
45
50
55
60
65
70
75
80
83
YEAR

FIGURE 2

WATERBORNE DISEASE
1920–1983